Case reports

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Dissimilar atrial rhythms[1]
A patient with interatrial block

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A patient with type A Wolff-Parkinson-White syndrome and prolonged interatrial conduction intervals developed atrial flutter during the course of an electrophysiological study. The atrial flutter blocked along the left-to-right conduction pathways in a Wenckebach pattern. The dissimilar atrial rhythms of right atrial tachycardia and left atrial flutter evolved as the interatrial block increased to 2:1 conduction.

Dissimilar atrial rhythms in man have been reported infrequently since Schrumpf's first description (Schrumpf, 1920). The forms of dissimilar atrial rhythms reported to date include dissociated regular rhythms of each atrium (Schrumpf, 1920; Bay and Adams, 1932; Scherf, 1955; Marques, 1958; Abarquez and La Due, 1961; Cohen and Scherf, 1965; Chung, 1971; Sivertssen and Jörgensen, 1973; Clark and Douglas, 1973; Scott and Finnegan, 1975), sinus rhythm with a segment of the atria in tachycardia (Chung, 1971), flutter (Chung, 1971; Dayem *et al.*, 1972), or fibrillation (Cohen and Scherf, 1965; Chung, 1969, 1971), and a rapid regular rhythm (tachycardia or flutter) of one atrium with simultaneous fibrillation of the other atrium (Zipes and Dejoseph, 1973; Leier and Schaal, 1975). Atrial standstill with flutter in another segment of the atria has also been reported to occur in man (Zipes and Dejoseph, 1973; Leier and Schaal, 1975; Wu *et al.*, 1975). We report a patient with left atrial flutter and right atrial tachycardia which occurred in the setting of 2:1 interatrial block and interatrial Wenckebach block.

Case report

The patient was a 66-year-old man with type A Wolff-Parkinson-White (WPW) syndrome and a 60-year history of intermittent tachycardia. Self-administered carotid massage consistently interrupted the tachyarrhythmia. Over the preceding 5 years, the episodes of tachycardia had increased in frequency and duration and were frequently accompanied by shortness of breath. No abnormality was found on physical examination. The electrocardiogram showed type A WPW pre-excitation. Other laboratory tests gave normal results.

Methods

A bipolar catheter was placed in the oesophagus for recording left atrial activity. Two bipolar catheters were placed in the high right atrium, one for right atrial pacing and the other for recording a high right atrial electrogram. A six-pole catheter was placed across the tricuspid valve for bundle of His and low right atrial recordings. Standard leads I, II, III, left atrial, and intracavitary right heart electrograms were recorded simultaneously on a multichannel oscilloscopic recorder (Electronics for Medicine DR-12) at paper speeds of 50 and 100 mm/s. Electrical stimuli (2 × threshold, 2 ms duration) were introduced by a pulse stimulator (Grass Instrument Model S 88, Quincy, Massachusetts).

Results

Baseline electrophysiological data obtained included a high to low right atrium conduction interval of 40 ms (normal < 48 ms—Leier *et al.*, 1976), a prolonged right to left atrial conduction interval of 70 ms (normal < 60 ms—Leier *et al.*, 1976), an AH interval of 90 ms, and an HV interval (to onset of delta wave) measuring 16 ms. Sinoatrial conduction times and corrected sinoatrial recovery times were found to be normal at 110 to 125 ms and 110 to 150 ms, respectively (Engel *et al.*, 1976). The effective refractory periods of the atrioventricular node and of the accessory pathway in sinus rhythm

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were 260 ms and 280 ms, respectively. The high to low right atrium and the right to left atrial conduction times increased above baseline values with atrial pacing (up to pacing rates of 160/min) and with atrial premature stimuli, but these manoeuvres did not elicit atrial Wenckebach block or any other form of intra- or interatrial block.

Placement of an atrial premature stimulus in the right atrium at an A_1-S_1 interval of 280 ms consistently initiated an episode of supraventricular tachycardia with atrial and ventricular rates of 136/min. These episodes were terminated with placement of an atrial premature stimulus in the right atrium at an A_1-S_1 interval of 250 ms. The atrioventricular node was refractory at this interval and the loop was interrupted. Propranolol 2 mg was administered intravenously over two minutes to determine its effectiveness in suppressing the tachyarrhythmia. Approximately seven minutes after the propranolol administration, an atrial premature stimulus with a 280 ms A_1-S_1 interval again initiated an atrial tachycardia at 136/minute. An additional atrial premature stimulus with an A_1-S_1 interval of 280 ms changed the atrial tachycardia to atrial flutter with an atrial rate of 280/minute. The left atrial rhythm continued as atrial flutter at 280/minute, but the right atrial depolarisations became irregular and slower with variable conduction time intervals between the left and right atria (Fig. 1A and B). Fig. 1B shows a recurrent pattern of pro-

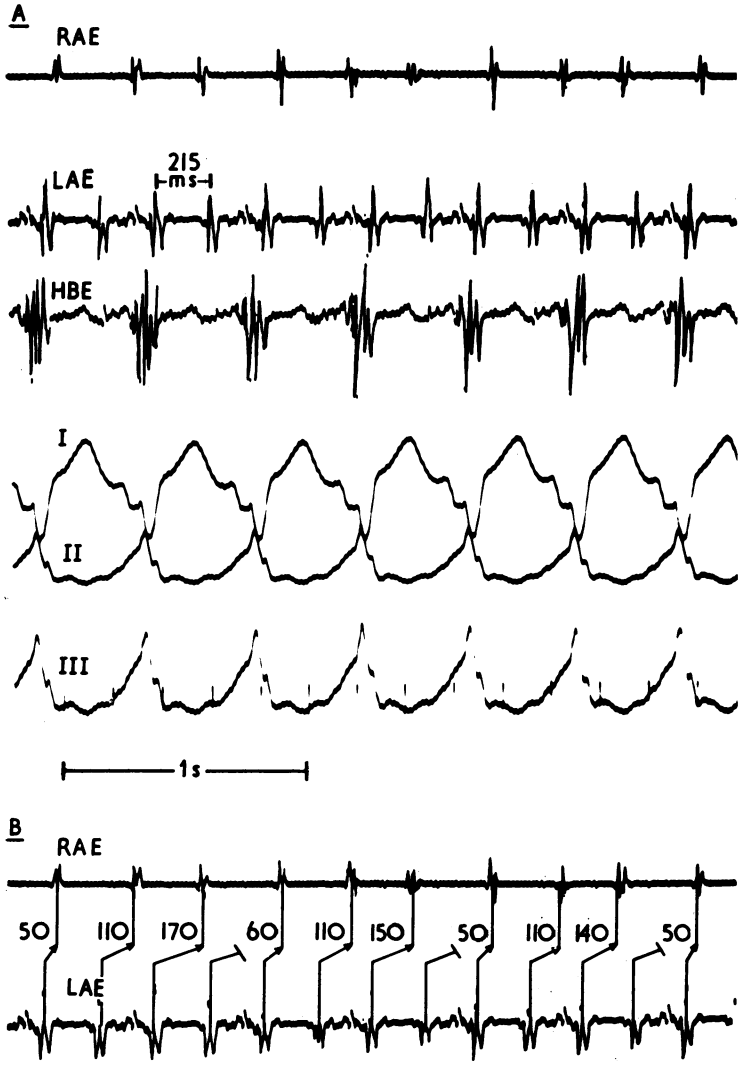

Fig. 1 *(A) Recordings taken during the atrial flutter with left-to-right Wenckebach. The left atrial rate is 280/min (AA interval=215 ms). The ventricular rate is regular with a constant RR interval of 430 ms. (B) A sketch of the atrial events recorded in Fig. 1A shows the gradual prolongation of the left to right atrial conduction interval (ms) until a left atrial complex is not conducted to the right. RAE, right atrial electrogram; LAE, left atrial electrogram; HBE, His bundle electrogram.*

gressive lengthening of the conduction interval from the left to the right atrium with the eventual loss of a right atrial complex. The interval between the right atrial complexes shortened until a left atrial impulse was not conducted to the right atrium. It is noteworthy that the ventricular rate did not vary during the development of the interatrial block, indicating that the left atrium was activating the ventricles with 2:1 conduction and thus providing further evidence that the accessory pathway is left sided (type A WPW). The 4:3 interatrial block gradually changed into a 2:1 block, resulting in dissimilar atrial rhythms of left atrial flutter at 280/minute and right atrial tachycardia of 140/minute (Fig. 2). The 2:1 interatrial block intermittently reverted to 3:2 or 4:3 interatrial block for brief periods. Several minutes later, the dissimilar rhythms gave way to regular sinus rhythm.

Discussion

This patient illustrates another form of dissimilar atrial rhythms with flutter of one atrium and atrial tachycardia of the other atrium. During the interatrial block the scalar electrocardiogram gave no information about atrial events occurring at the time. This emphasises the importance of recording simultaneous electrograms from each atrium to demonstrate atrial events in patients with atrial arrhythmias.

The biatrial recordings during atrial flutter showed a recurrent pattern of gradual prolongation of the left to right conduction time until a left atrial impulse was not conducted to the right atrium. Concurrently, the right atrial A wave intervals progressively shortened until the dropped right atrial beat (Fig. 1). This pattern meets the criterion

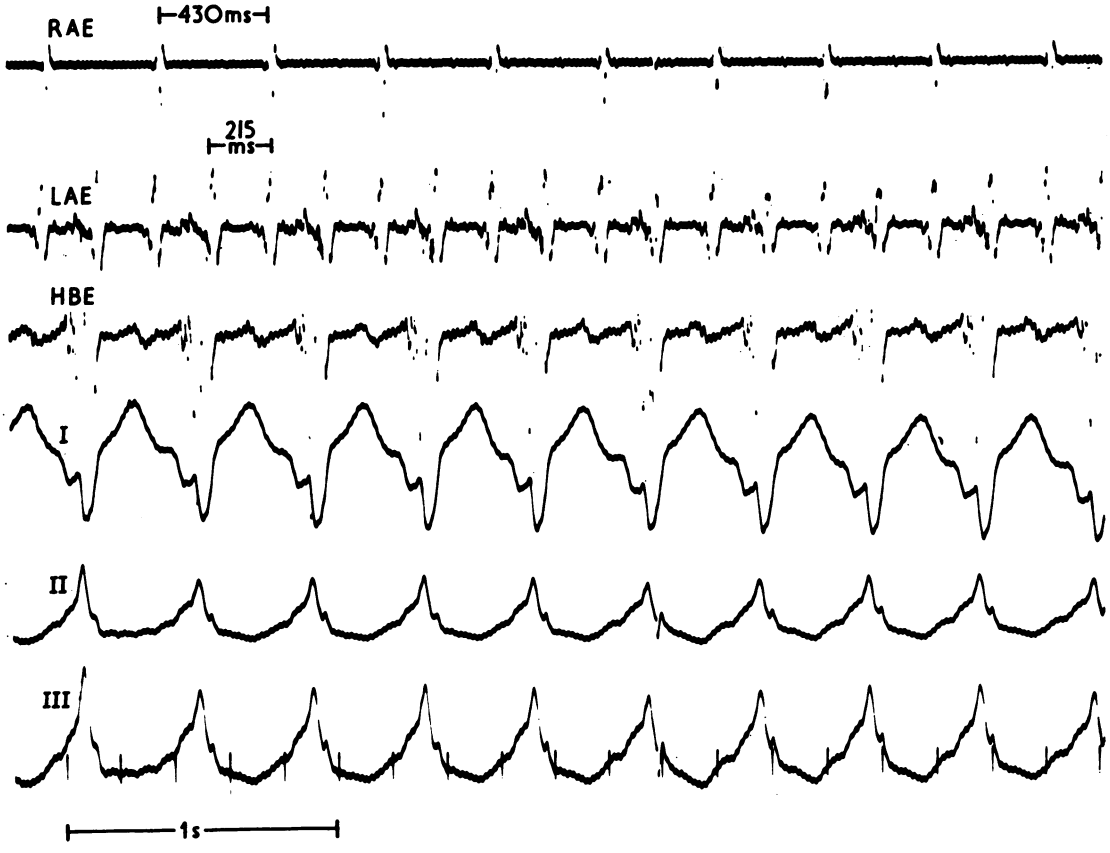

Fig. 2 *The atrial Wenckebach evolves into a 2:1 left-to-right conduction block resulting in right atrial rate of 140/min (AA interval=430 ms) in the presence of a left atrial rate of 280/min (AA interval=215 ms). For abbreviations, see Fig. 1.*

of Wenckebach conduction block. The 4:3 block gradually evolved into a 2:1 block resulting in a right atrial rate of 140/minute during left atrial flutter at a rate of 280/minute. The exact location of the atrial conduction block cannot be determined from the recordings. It is obvious that the block is located somewhere between the right and left atrial recording electrodes. We cannot assume that the atrial septum or Bachman's bundle are the regions of interatrial block since we did not explore the atria with the recording electrodes during the tachyarrhythmia. Though the low right atrial recording was not optimal in quality, most low right atrial deflections occurred in conjunction with the high right atrial deflections, indicating that most of the right atrium displayed the same rhythm.

Lewis *et al.* (1918–1920) produced intra-atrial Wenckebach block in the dog with rapid atrial pacing (369/minute). The atrial Wenckebach phenomenon has also been shown in isolated atrial muscle strips submitted to various physical and pharmacological manoeuvres (Lewis and Drury, 1923; Drury and Andrus, 1924; Drury and Regnier, 1927–1929). Early descriptions of the atrial Wenckebach phenomenon in man were made by Phibbs (1963), Scherf and Cohen (1964), and Schamroth (1967). Naruka *et al.* (1971, 1972) documented the occurrence of intra-atrial Wenckebach block in man using intracardiac recordings. They reported patients with abnormal high right to low right atrial conduction times who showed progressive prolongation of this interval during atrial pacing. Intra-atrial Wenckebach block was also documented with intracardiac recordings during atrial pacing (Castellanos *et al.*, 1972). One of their patients also showed progressive prolongation of the right to left atrial conduction interval indicating the presence of right to left interatrial Wenckebach block as well. The baseline prolonged interatrial conduction intervals noted in our patient lengthened with atrial pacing, though right to left atrial Wenckebach block did not develop with pacing rates up to 160/minute. The development of atrial flutter at a rate of 280/minute evoked block in the form of the Wenckebach phenomenon in the conduction pathways from the left to the right atrium. We did not pace the right atrium at rates between 160 and 280/minute to determine if the Wenckebach phenomenon would have occurred from the right to the left atrium.

Atrial conduction block has been shown to be secondary to various anatomical, physiological, and pharmacological factors (Fredericq, 1901; Lewis *et al.*, 1918–1920; Lewis and Drury, 1923; Drury and Andrus, 1924; Drury and Regnier, 1927–1929; Scherf and Siedeck, 1934; Legato *et al.*, 1974; Wu *et al.*, 1975). The patient described here had atrial conduction disease, with prolonged interatrial conduction times during sinus rhythm. The patient was not taking any drugs before the study and thus may have had atrial muscle pathology, as reported by Legato *et al.* (1974). The propranolol administered 7 to 9 minutes before the development of the dissimilar rhythms may have contributed to the atrial conduction disturbance as well as possibly increasing the refractory period (Seides *et al.*, 1974) of the atrial conduction pathways or of most of the right atrium. The right atrial rate was then limited to 140 to 210/minute in the presence of a left atrial rate of 280/minute.

References

Abarquez, R. F., and La Due, J. S. (1961). Atrial dissociation. *American Journal of Cardiology*, **8**, 448–452.

Bay, E. B., and Adams, W. (1932). Possible intranodal block. A report of cases. *American Heart Journal*, **7**, 759–766.

Castellanos, A., Iyengar, R., Agha, A. S., and Castillo, C. A. (1972). Wenckebach phenomenon within the atria. *British Heart Journal*, **34**, 1121–1126.

Chung, E. K. (1969). Atrial dissociation due to unilateral atrial fibrillation. *Journal of Electrocardiology*, **2**, 373–376.

Chung, E. K. (1971). A reappraisal of atrial dissociation. *American Journal of Cardiology*, **28**, 111–117.

Clark, W. H., and Douglas, J. E. (1973). Atrial dissociation—fact or artifact? *Journal of Electrocardiology*, **6**, 373–377.

Cohen, J., and Scherf, D. (1965). Complete interatrial and intra-atrial block (atrial dissociation). *American Heart Journal*, **70**, 23–34.

Dayem, M. K. A., Aytan, N., and Argano, B. J. (1972). Multiple atrial arrhythmias in intra-atrial and interatrial block. *Journal of Electrocardiology*, **5**, 281–288.

Drury, A. N., and Andrus, E. C. (1924). The influence of hydrogen-ion concentration upon conduction in the auricle of the perfused mammalian heart. *Heart*, **11**, 389–403.

Drury, A. N., and Regnier, M. (1927–1929). Observations upon conduction in the mammalian heart. Auricular conduction. *Heart*, **14**, 263–279.

Engel, T. R., Bond, R. C., and Schaal, S. F. (1976). First-degree sinoatrial heart block: sinoatrial block in the sick-sinus syndrome. *American Heart Journal*, **91**, 303–310.

Fredericq, L. (1901). Sur les pulsation de la veine cave superieure et des oreillettes du coeur chez le chien. *Bulletin de l'Académie Royale de Médecine de Belgique. Classe des Sciences*, **3**, 126.

Legato, M. J., Bull, M. B., and Ferrer, M. I. (1974). Atrial ultrastructure in patients with fixed intra-atrial block. *Chest*, **65**, 252–261.

Leier, C. V., and Schaal, S. F. (1975). Biatrial electrograms in the diagnosis of dissimilar atrial rhythms. *Circulation*, **52**, Suppl. II, 207.

Leier, C. V., Meacham, J. A., and Schaal, S. F. (1976). Atrial flutter: abnormal atrial conduction and sinus node dysfunction (abstract). *Clinical Research*, **24**, 226A.

Lewis, T., and Drury, A. N. (1923). The effect of vagal stimulation on intra-auricular block produced by pressure or cooling. *Heart* **10**, 179–195.

Lewis, T., Feil, H. S., and Stroud, W. D. (1918–1920). Observations upon flutter and fibrillation. Part III. Some effects of rhythmic stimulation of the auricle. *Heart*, **7**, 247–272.

Marques, M. G. (1958). Atrial dissociation. *British Heart Journal*, **20**, 335–340.

Narula, O. S., Runge, M., and Samet, P. (1972). Second-degree Wenckebach type AV block due to block within the atrium. *British Heart Journal,* **34,** 1127–1136.

Narula, O. S., Scherlag, B. J., Samet, P., and Javier, R. P. (1971). Atrioventricular block. Localisation and classification by His bundle recordings. *American Journal of Medicine,* **50,** 146–165.

Phibbs, B. (1963). Paroxysmal atrial tachycardia with block around the ectopic pacemaker. *Circulation,* **28,** 949–950.

Schamroth, L. (1967). The theory and mechanism of the Wenckebach phenomenon. *South African Medical Journal,* **41,** 827–832.

Scherf, D. (1955). The atrial arrhythmias. *New England Journal of Medicine,* **252,** 928–933.

Scherf, D., and Cohen, J. (1964). *The Atrioventricular Node and Selected Cardiac Arrhythmias,* p. 327. Grune and Stratton, New York.

Scherf, D., and Siedeck, H. (1934). Uber Block zwischen beiden Vorhöfen, *Zeitschrift für klinische Medizin,* **127,** 77–88.

Schrumpf, P. (1920). De l'Interference de deux rythmes sinusaux; preuve due dualisme du nodule de Keith. *Archives des Maladies du Coeur et des Vaisseaux,* **13,** 168–173.

Scott, M. E., and Finnegan, O. C. (1975). Atrial dissociation. *British Heart Journal,* **37,** 539–542.

Seides, S. F., Josephson, M. E., Batsford, W. P., Weisfogel, G. M., Lau, S. H., and Damato, A. N. (1974). The electrophysiology of propranolol in man. *American Heart Journal,* **88,** 733–741.

Sivertssen, E., and Jörgensen, L. (1973). Atrial dissociation. *American Heart Journal,* **85,** 103–107.

Wu, D., Denes, P., Amat y Leon, F., Chhablani, R. C., and Rosen, K. M. (1975). Limitations of the surface electrocardiogram in diagnosis of atrial arrhythmias. *American Journal of Cardiology,* **36,** 91–97.

Zipes, D. P., and Dejoseph, R. L. (1973). Dissimilar atrial rhythms in man and dog. *American Journal of Cardiology,* **32,** 618–628.

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